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## ORIGINAL COMMUNICATION

# The effect of guar gum addition to a semisolid meal on appetite related to blood glucose, in dieting men

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**Objective:** To investigate whether addition of modified guar gum (GG) to a low-energy semisolid meal might be effective on appetite by modifying the response of blood glucose and other blood parameters.

**Design:** Three intervention periods of 2 weeks each, separated by washout periods of 4 weeks. Randomized and cross-over design.

**Subjects:** Fifteen overweight male subjects (mean  $\pm$  s.d.; age,  $44 \pm 9$  y; body mass index,  $28.6 \pm 1.8$  kg/m<sup>2</sup>).

**Intervention:** Subjects consumed a low-energy diet divided over three times a day, consisting of a semisolid meal with (SSM+) or without (SSM) addition of 2.5 g GG, or a solid meal (SM) with the same energy content (947 kJ) and macronutrient composition, plus a dinner of the subject's own choice. At the end of each intervention, time and number of meal initiations, dynamics of blood glucose and other blood parameters, and appetite ratings such as hunger and satiety were determined in a time-blinded situation.

**Results:** The changes in blood glucose from meal initiation to blood glucose peak and from peak to nadir were smaller with SSM+ and SM compared to SSM. Satiety before the third meal was higher with SSM+ and SM compared to SSM ( $P < 0.01$ ). Meal pattern, general appetite and total energy intake were similar for all treatments.

**Conclusions:** We conclude that, similar to SM, SSM+ resulted in a more moderate change in blood glucose compared to SSM and positively affected satiety before the third meal, while general appetite, total energy intake and meal pattern did not differ. *European Journal of Clinical Nutrition* (2002) 56, 771–778. doi:10.1038/sj.ejcn.1601407

**Keywords:** guar gum; blood glucose; intermeal interval; appetite; satiety; food intake regulation; meal pattern

### Introduction

In the treatment of obesity, long-term reduction of energy intake is one of the cornerstones of therapy. Low-energy diets have been shown to be successful in the reduction of body weight in the short term, but in the long term decreased compliance and habituation result in unsuccessful weight loss or in weight regain (Doucet *et al*, 2000). Reduced hunger and increased satiety feelings may improve compliance to an energy-restricted diet (Astrup *et al*, 1990). There-

fore, identification of substances that improve or sustain satiety during energy restriction is needed for the treatment of obesity.

The effects of dietary fibers on satiety, energy intake and/or body weight have been extensively investigated (Stevens, 1988; Blundell & Burley, 1987; Burley & Blundell, 1990). Dietary fibers such as guar gum (GG) were found to effectively increase fullness and satiety resulting in reduced energy intake (Evans & Miller, 1975; Pasman *et al*, 1997), eg by decreasing the rate of gastric emptying and small intestinal transit (Harju, 1985; Wilmshurst & Crawley, 1980; Blackburn *et al*, 1984a). However, other more recent studies found no effects of GG supplementation on gastric emptying and/or intestinal transit rate (Rydning *et al*, 1985; Lavin & Read, 1995; Meier *et al*, 1993). It has also been observed that GG ingestion may modify glucose absorption (Blackburn *et al*, 1984b) resulting in a prolonged feeling of satiety (Leeds, 1987).

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Controversial findings have been shown on the effects of different types of dietary fibers along with an energy-restricted diet on energy intake and/or body weight loss (Hylander & Rössner, 1983; Solum *et al*, 1987; Rössner *et al*, 1987, 1988; Rytting *et al*, 1989; Astrup *et al*, 1990). To our knowledge, only a few studies have investigated the effects of GG supplementation during energy restriction on energy intake, hunger and satiety (Pasman *et al*, 1997; Heini *et al*, 1998). Pasman *et al*, (1997) found a hunger-reducing effect of GG during the energy-restricted condition, suggesting that GG might be useful in the treatment of obesity, by increasing the compliance to a low-energy diet. However, Heini *et al* (1998) did not confirm this effect of GG.

In a larger study, we investigated the effects of GG addition to a low-energy semisolid meal on appetite and body weight loss during a 2-week intervention. It was shown that GG addition to a low-energy semisolid meal prevented an increase in appetite, hunger and desire to eat, while this increase was present in the unsupplemented semisolid meal and in a low-energy solid meal (Kovacs *et al*, 2001).

The aim of the present study was to investigate whether this satiating effect of GG addition to a low-energy semisolid meal is mediated by modifying the response of blood glucose and/or other blood parameters, also in comparison to the effects of a similar but solid meal. We hypothesized that addition of GG to the semisolid meal would result in a more moderate change in blood glucose and insulin, similar to the solid meal, and that this would affect appetite parameters.

## Subjects and methods

### Subjects

Fifteen overweight male subjects participated in this study. The subjects were recruited by advertisements in local newspapers, in which we asked for moderately obese male subjects who wanted to participate in a study on the effects of different low-energy diets on appetite, food intake and body weight. The subjects were a subsample of the study mentioned before that is described in Kovacs *et al* (2001). Selection for this study with a more intensive protocol took place according to health criteria (fasting blood glucose <6.2 mmol/l; blood pressure <95 mmHg (diastolic) and <160 mmHg (systolic), no diabetes, no cardiovascular diseases, and no medical treatment) and body weight criteria (body mass index: 25–30 kg/m<sup>2</sup>). Baseline characteristics of the subjects are presented in Table 1. The subjects did not use fiber supplements regularly before the study. The nature and risks of the experimental procedure were explained to the subjects, and all subjects gave their written informed consent. The study was approved by the Ethical Committee of Maastricht University.

### Experimental design

The experiment had a randomized and cross-over design. The experimental design consisted of three treatments, each

**Table 1** Subject characteristics at baseline

	Mean $\pm$ s.d.	Range
Age (y)	43.7 $\pm$ 9.3	29–56
Height (m)	1.77 $\pm$ 0.06	1.65–1.88
Weight (kg)	89.2 $\pm$ 7.7	79.6–102.7
Body mass index (kg/m <sup>2</sup> )	28.6 $\pm$ 1.8	25.9–32.4
Waist circumference (cm)	98 $\pm$ 6	91–108
Hip circumference (cm)	104 $\pm$ 5	96–112
Waist–hip ratio	0.94 $\pm$ 0.04	0.85–1.01
Body fat (%)	30.9 $\pm$ 1.1	23.3–38.5
Systolic blood pressure (mmHg)	149 $\pm$ 23	116–206
Diastolic blood pressure (mmHg)	91 $\pm$ 11	77–106
Blood glucose (mmol/l)	5.4 $\pm$ 0.2	4.86–5.65
F1 (cognitive restraint)	4.0 $\pm$ 2.8	0–9
F2 (disinhibition)	3.9 $\pm$ 1.4	1–5
F3 (hunger)	3.5 $\pm$ 2.1	0–8
Herman Polivy restraint	13.7 $\pm$ 4.5	6–20

n = 15 men. F1–F3, factors 1–3 of the Three Factor Eating Questionnaire. For Herman Polivy restraint (normal <15) and F1–F3 scores (normal <9) a higher value indicates more restraint, disinhibition, hunger.

consisting of a 2 week low-energy diet and a subsequent time-blinded test, separated by a 4 week washout period (Figure 1; Kovacs *et al*, 2001).

Daily during the intervention periods, the subjects consumed a low-energy meal for breakfast, lunch and as a 4 o'clock snack. In addition, for lunch, subjects were allowed to eat a fruit (eg apple, pear, orange). For dinner, the subjects ingested what they liked, but they were instructed to drink no more than one glass of alcoholic beverage or soft drink. Subjects were instructed to refrain from snacks in between meals. During the day, the subjects were allowed to drink *ad libitum* water, coffee and tea (without sugar and milk). The low-energy meal consisted of a semisolid meal with (SSM+) or without (SSM) addition of 2.5 g of modified GG (Meyprofin<sup>®</sup> M-175, Meyhall AG, Kreuzlingen, Switzerland, a highly purified dietary galactomannan fiber which forms a gel approximately 5–7 min after adding water) or a solid meal (SM). A relatively low dosage of 2.5 g of GG was chosen because a higher dosage appeared not to affect gastrointestinal transit rate but to increase the viscosity of the semisolid meal, thus reducing palatability (van Nieuwenhoven *et al*, 2001). The semisolid meal was a pudding (Milical Crème, Laboratoires Diététique et Santé, Revel, France, 47 g dissolved in 200 ml water; energy, 947 kJ; protein, 17.9 g; carbohydrate, 21.0; fat, 7.9 g). It was offered in three flavors (vanilla, chocolate, caramel) and the subjects were allowed to choose the meals in the flavors they preferred. The solid meal consisted of bread with cheese, meat or fish, salad or vegetables with dressing, and had the same energy content and macronutrient composition as the semisolid meal. The subjects prepared the solid meal themselves using our specific instructions. These instructions indicated the type of food and the amount that the subjects were allowed to

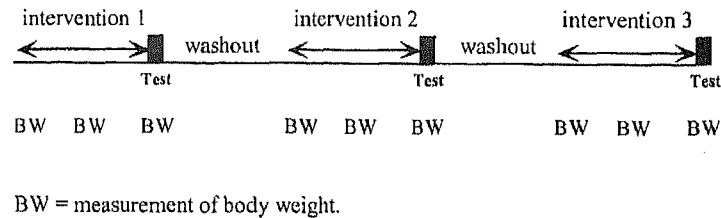


Figure 1 Experimental design.

consume. Compliance to the dietary instructions was controlled using food intake diaries (Kovacs *et al*, 2001).

At the end of each intervention, the subjects reported to the laboratory between 8:00 h and 9:00 h, following a 10 h overnight fast, for a testing day (Figure 2). Testing started at different times between 8:00 and 9:30 h that were randomly chosen for the purpose of time blinding. During the test, subjects were housed in a room and isolated from time and food cues to eliminate habitual (time-determined) meal patterns, enabling the investigators to observe meal responses to mainly physiological cues. Television, video, radio, watches and clocks were not in the room, and research staff refrained from making time-related statements. With exception of the investigators and the subject, no other people were allowed in the room. Subjects sat on a bed and were allowed to read or write during the test. They were not allowed to sleep. Subjects could verbally request a meal (= meal initiation) at any time. According to the randomly assigned treatment, subjects consumed, upon the first, second and third meal request (breakfast, lunch and snack, respectively), the same low-energy meal (SSM+, SSM or SM) they received during the preceding 2 weeks. The semisolid meal consisted of a portion of pudding and the solid meal consisted of white bread (50 g), low-fat margarine (7 g), ham (50 g), and tomato (50 g). During the day, water, coffee and tea (without sugar or milk) were offered *ad libitum*. Then, upon their fourth meal request (dinner), they ingested *ad libitum* a standardized meal (950 g pasta with tomato sauce; per 100 g: energy, 553 kJ; protein, 5 g; carbohydrate, 19.2 g; fat, 3.9 g). The dinner was offered in another room from the universal eating monitor (Kissileff *et al*, 1980; Westterp-Plantenga *et al*, 1990). The subjects were allowed to drink water during the meal. Testing was stopped at the subject's fourth meal request or at 18:00 h if the subject had not

requested a third or fourth meal yet. If the subject requested the third meal after 17:00 h, testing was stopped at that time point. Upon completion of the testing, subjects were asked to estimate the clock time in order to verify that they were blinded to the time of the day.

### Measurements

**Anthropometry.** Body weight (BW) was measured during screening, at the beginning, after 1 week and at the end of each intervention period on a digital balance (Seca, model 707, Hamburg, Germany; weighing accuracy of 0.1 kg) with subjects in underwear, in a fasted state, and after voiding their bladder. Height was measured during screening using a wall-mounted stadiometer (Seca, model 220, Hamburg, Germany). The body mass index was calculated by  $BW/height^2$  ( $kg/m^2$ ).

The distribution of fat was determined during screening by measuring the waist and hip circumferences and calculation of the waist-hip ratio (WHR). The waist circumference was measured at the site of the smallest circumference between the rib cage and the iliac crest, with the subjects in a standing position. The hip circumference was measured at the side of the largest circumference between the waist and the thighs. The WHR was calculated by dividing the waist circumference by the hip circumference.

Body composition was measured at baseline using the deuterium ( $^2H_2O$ ) dilution technique (Schoeller *et al*, 1980). The dilution of the deuterium isotope is a measure for total body water (TBW; van Marken Lichtenbelt *et al*, 1994). Subjects were asked to collect a urine sample in the evening just before drinking a weighed amount of deuterium-enriched water. After ingestion of the deuterium-enriched water, no further fluid or food consumption was permitted.

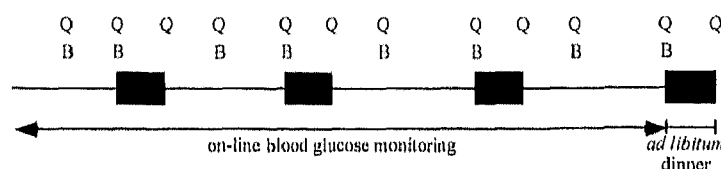


Figure 2 Design during testing. Solid rectangles = meal initiation; Q = questionnaires on appetite ratings; B = blood sampling.

Ten hours later a second urine sample (second voiding) was collected. Deuterium concentration in the urine samples was measured using an isotope ratio mass spectrometer (Micro-mass Optima, Manchester, UK). TBW was obtained by dividing the measured deuterium dilution space by 1.04 (Schoeller *et al*, 1980). Fat-free mass (FFM) was calculated by dividing the TBW by the hydration factor 0.73. By subtracting FFM from BW, fat mass (FM) was obtained. FM expressed as a percentage of BW revealed body fat percentage.

**Attitude towards eating.** Attitude towards eating with respect to dietary restraint was determined during screening using a validated Dutch translation of the Three Factor Eating Behavior Questionnaire (TFEQ; Stunkard & Messick, 1985; Westerterp-Plantenga *et al*, 1999). Cognitive restrained and unrestrained eating behavior (factor 1), emotional eating and disinhibition (factor 2) and the subjective feelings of hunger (factor 3) were scored. Body weight concern and chronic dieting behavior were investigated with the Herman Polivy questionnaire (HP; Herman & Polivy, 1980).

**Blood parameters.** Systolic and diastolic blood pressures were measured during screening by an automatic blood pressure monitor (Omron 705 CP, Omron Healthcare GmbH, Hamburg, Germany). A fasted blood sample (2 ml) was obtained and mixed with EDTA to prevent clotting. Plasma was obtained by centrifugation and then stored at  $-20^{\circ}\text{C}$  until analysis of glucose by a hexokinase method (Cobas Bio, Roche Diagnostics, Hoffmann-La Roche, Basel, Switzerland).

#### Testing day

**On-line blood glucose monitoring.** After the subject was comfortably settled, an 18-gauge, 5 cm angiocath was placed in the antecubital vein of the non-dominant arm. The blood withdrawal end of a specially modified, 2.5 m long double-lumen catheter (MTB Medizintechnik, Amstetten, Germany) was fitted into the angiocath. The catheter was continuously heparinized by pumping a sterile heparin-saline solution (500–5000 U/ml) at a rate of approximately 25  $\mu\text{l}/\text{min}$  through the distal lumen of the catheter to the tip of the cannula. The blood and heparin-saline solution mixture was continuously withdrawn through the proximal lumen of the cannula at a rate of approximately 50  $\mu\text{l}/\text{min}$  (blood withdrawal rate = 25  $\mu\text{l}/\text{min}$ ). The blood and heparin-saline solution mixture was mixed with a heparinized phosphate buffer, at a 1:10 ratio, and continuously infused into a sample chamber of a glucose analyzer (Model 23A, Yellow Springs Instrument Co., Yellow Springs, OH, USA). Approximately 15 ml of blood were withdrawn on the testing day. The transit time of this continuous sampling was approximately 4 min, which was timed for each catheter, and accounted for in the data analysis. Sampling occurred at a rate of 10 times per min, and analogue data were amplified,

digitized, interfaced (Data Translation Interface Board, model 1028), and displayed continuously on a computer monitor. This monitor was not visible to the subjects. One minute averages of blood glucose levels over time were plotted for each subject's testing day. For 30 min prior to the insertion of the catheter into the subjects, and following the completion of testing, the system was calibrated using a bag of sterile saline with a known concentration of glucose added. This calibration was done using the same blood-withdrawal cannula that was used in the subject that day (Melanson *et al*, 1999a–c).

**Blood parameters.** An additional 20-gauge, 3.2 cm angiocath was placed in the antecubital vein of the other arm for occasional blood sampling. At baseline, just before and 35 min after breakfast, lunch and snack and just before dinner, a 9 ml blood sample was collected into an EDTA-containing tube to prevent clotting. Plasma was obtained by centrifugation ( $4^{\circ}\text{C}$ , 3000 rpm, 10 min) and stored at  $-80^{\circ}\text{C}$  until analysis of glucose by the hexokinase method (Roche Diagnostics, Hoffmann-La Roche, Basel, Switzerland), free fatty acids by the ACS-ACOD method (Wako Chemicals, Neuss, Germany), glycerol by the glycerolkinase-lipase method (Boehringer, Mannheim),  $\beta$ -hydroxybutyrate by the method of Moore *et al* (1982) using a semi-automated centrifugal spectrophotometer (Cobas Fara, Roche Diagnostics), and insulin with the 1235 AutoDELFIA automatic immunoassay system (AutoDELFIA Insulin kit B080-101, WallacOy, Turku, Finland).

**Appetite ratings.** Appetite ratings (ie ratings of hunger, satiety, fullness, desire to eat, appetite, anticipated food intake and thirst) were scored on 100 mm visual analog scales (VAS) anchored with 'not at all' and 'extremely'. Questionnaires were completed at nine fixed time points, respectively at baseline, before, immediately after breakfast, lunch, snack, as well as before and immediately after dinner. Appetite ratings, corrected for the subject's minimum score, were expressed as area under the curve (AUC) over 10 h. Questionnaires were completed at three further time points (35 min after breakfast, lunch, and snacks) for comparison with blood parameters.

#### Statistics

Data are presented as mean  $\pm$  standard error (s.e.). Differences between the treatments were determined by analysis of variance for repeated measures (ANOVA) and Sheffe-*F* post hoc test (Statview SE Graphics<sup>®</sup>). Pearson correlation coefficients, *r*, were calculated to determine the relationship between results from blood sampling and outcomes such as appetite ratings and intermeal interval. This was performed for the three treatments separately, as well as for all the test days combined. The level of significance was set at  $P < 0.05$ .

## Results

BW loss during 2 weeks of intervention was  $2.5 \pm 0.4$ ,  $1.8 \pm 0.3$  and  $1.8 \pm 0.4$  kg during SSM+, SSM and SM, respectively, with no significant differences between treatments. The subjects were in negative energy balance on the testing day.

The subject's estimation of clock time at the end of the testing day ranged from  $-96$  to  $+198$  min, verifying that the subjects were blinded to the time of the day. During each test two to four meal initiations occurred, but there was no difference in the number of meal initiations between treatments (SSM+,  $3.1 \pm 0.2$ ; SSM,  $3.2 \pm 0.2$ ; SM,  $3.0 \pm 0.2$ ). Figure 3 depicts parameters calculated from the on-line blood glucose monitoring curves. Average baseline blood glucose was higher in treatment SM compared to SSM ( $P < 0.05$ ), with values from treatment SSM+ intermediate. The time at which meal initiation, glucose peak or subsequent glucose nadir occurred in relation to breakfast, lunch or snack was similar for all treatments. However, time from meal initiation to glucose peak was longer in treatment SM compared to SSM and SSM+ ( $P < 0.01$ ). Glucose at meal initiation was higher during treatment SM compared to SSM ( $P < 0.05$ ). Glucose at peak was similar for all treatments, but glucose at the subsequent nadir was higher in treatment SM compared to SSM and SSM+ ( $P < 0.001$ ). The 28 declines in blood glucose observed during treatment SSM+ constituted a  $34.5 \pm 3.2$  mg/dl decrease (30%), which followed a rise in blood glucose induced by meal ingestion ( $29.2 \pm 2.3$  mg/dl, 34.2%). The 32 declines in blood glucose observed during treatment SSM constituted a  $48.5 \pm 5.5$  mg/dl decrease (39%), which followed a rise in blood glucose induced by meal ingestion ( $41.4 \pm 4.9$  mg/dl, 49%). The 26 declines in blood glucose observed during treatment SM constituted a  $31.6 \pm 3.0$  mg/dl decrease (26%), which followed a rise in blood glucose induced by meal ingestion ( $31.5 \pm 2.5$  mg/dl, 35%). Increase in blood glucose from meal initiation to peak was smaller in treatment SSM+ compared to SSM ( $P < 0.05$ ).

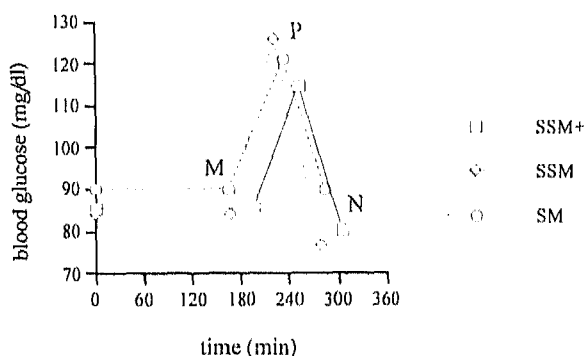


Figure 3 Data obtained from the on-line glucose monitoring curves. Values are mean. SSM+ = semisolid meal with guar gum; SSM = semisolid meal; SM = solid meal; M = meal initiation; P = blood glucose peak; N = blood glucose nadir.

Decrease in blood glucose from peak to nadir was significantly smaller in SSM+ and SM compared to SSM ( $P < 0.05$ ).

Plasma fasting levels of glucose, insulin, free fatty acids, glycerol and  $\beta$ -hydroxybutyrate during the testing day were not different between treatments (Table 2).

Plasma glucose concentration increased as a result of meal consumption and then decreased until next meal consumption. Plasma glucose concentration before lunch was higher during SM treatment compared to SSM ( $P < 0.05$ ). However, mean plasma glucose concentration over time was similar for all treatments. Plasma insulin concentration increased after ingestion of a meal and decreased thereafter (Figure 4). Plasma insulin concentration during treatment SSM was lower compared to SM before lunch ( $P < 0.01$ ), and higher 35 min after both lunch and snack ( $P < 0.05$ ). However, mean plasma insulin concentration over time was similar for all treatments.

There were no significant differences in plasma free fatty acids, glycerol and  $\beta$ -hydroxybutyrate concentrations between treatments at any time points and over time (data not shown).

Plasma glucose and insulin concentrations were negatively correlated with hunger ( $r = -0.50$  and  $r = -0.44$ ,

Table 2 Fasting plasma parameters

	SSM+	SSM	SM	P
Glucose (mmol/l)	$5.3 \pm 0.1$	$5.2 \pm 0.1$	$5.3 \pm 0.1$	NS
Insulin (U/l)	$8.3 \pm 0.6$	$8.0 \pm 0.7$	$9.3 \pm 0.7$	NS
Free fatty acids ( $\mu$ mol/l)	$430 \pm 38$	$377 \pm 34$	$417 \pm 51$	NS
Glycerol ( $\mu$ mol/l)	$70 \pm 7$	$67 \pm 7$	$80 \pm 11$	NS
$\beta$ -Hydroxybutyrate (mmol/l)	$271 \pm 34$	$219 \pm 27$	$284 \pm 62$	NS

Values are mean  $\pm$  s.e.

SSM+ = semisolid meal with guar gum; SSM = semisolid meal; SM = solid meal.

Statistical significance was determined by an analysis of variance for repeated measures (ANOVA). NS = not significant.

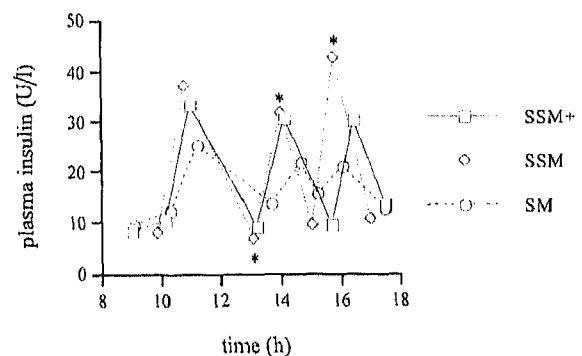


Figure 4 Plasma insulin concentration (U/l). Values are mean. SSM+ = semisolid meal with guar gum; SSM = semisolid meal; SM = solid meal. Statistical significance was determined by an analysis of variance for repeated measures (ANOVA). \*SSM significantly different from SM ( $P < 0.01$ ,  $P < 0.05$  and  $P < 0.05$ , respectively).

respectively;  $P < 0.001$ ) and positively correlated with satiety ( $r = 0.35$  and  $r = 0.43$ , respectively;  $P < 0.001$ ), measured at the same time points. Plasma glycerol concentrations were positively correlated with hunger ( $r = 0.21$ ;  $P < 0.001$ ) and negatively correlated with satiety ( $r = -0.23$ ;  $P < 0.001$ ). Plasma free fatty acid concentrations were positively correlated with hunger ( $r = 0.12$ ;  $P < 0.05$ ) but not with satiety, and plasma  $\beta$ -hydroxybutyrate concentrations were correlated with satiety ( $r = 0.14$ ;  $P < 0.05$ ) but not with hunger.

Meal frequency was similar for all treatments (SSM+,  $3.5 \pm 0.1$ ; SSM,  $3.5 \pm 0.1$ ; SM,  $3.3 \pm 0.1$ ). No significant difference in intermeal intervals between treatments was found from the first to the second meal (SSM+,  $190 \pm 15$  min; SSM,  $187 \pm 12$  min; SM  $202 \pm 15$  min), from the second to the third meal (SSM+,  $197 \pm 15$  min; SSM,  $180 \pm 10$  min; SM,  $180 \pm 11$  min), and from the third to the fourth meal (SSM+,  $110 \pm 10$  min,  $n = 7$ ; SSM,  $106 \pm 8$  min,  $n = 8$ ; SM,  $138 \pm 11$  min,  $n = 5$ ). There was no order effect for the number of meals or for the intermeal intervals.

The appetite ratings throughout the testing day, expressed as AUC, are shown in Table 3. The AUC of these parameters showed a similar trend and were not different between any of the three treatments. Satiety scores before the third meal (snack) were lower during SSM treatment compared to SSM+ and SM ( $P < 0.01$ ).

Energy intake during the *ad libitum* dinner did not differ between the treatments (SSM+,  $2.9 \pm 0.3$  MJ; SSM,  $2.9 \pm 0.3$  MJ; SM,  $2.9 \pm 0.3$  MJ). Total energy intake during the testing day amounted to  $5.7 \pm 0.3$  MJ for SSM+,  $5.6 \pm 0.3$  MJ for SSM and  $5.5 \pm 0.4$  MJ for SM (NS) and was significantly lower compared to baseline ( $10.2 \pm 0.6$  MJ,  $P < 0.001$ ; Kovacs *et al*, 2001).

## Discussion

The present study investigated the effect of guar gum addition to a low-energy semisolid meal on appetite related to blood glucose in overweight men consuming a low-energy diet. Effects were shown on satiety and blood glucose para-

meters, but not on total energy intake, meal pattern, or general appetite.

Satiety before the third meal was higher with SSM+ and SM compared to SSM. From continuous on-line glucose monitoring, it was observed that the increase in blood glucose from meal initiation to blood glucose peak and the decrease from blood glucose peak to nadir were more depressed with SSM+ and SM compared to SSM. A first explanation for the less marked changes in blood glucose and insulin with SSM+ compared to SSM could be that addition of GG decreases gastric emptying or intestinal transit time. However, in a previous study we observed that the addition of 2.5 to 4.5 g GG to the same low-energy semisolid meal had no effect on the gastric emptying rate and the rate of intestinal transit (van Nieuwenhoven *et al*, 2001). Thus other physiological processes are likely to be involved in reported satiating effects of GG supplementation (Pasman *et al*, 1997; Kovacs *et al*, 2001). GG may exert an action on glucose absorption by an impaired diffusion rate through the intestinal unstirred layer (Lembcke *et al*, 1984). This would lead to a prolonged period of glucose uptake in the intestine and consequently a prolonged influence on blood glucose and possibly on insulin levels. This, along with the fact that other nutrients may be absorbed at a lower rate, may affect satiety. Melanson *et al* (1999c) suggested that subjects who rapidly absorb carbohydrate into the blood stream feel satiated more rapidly, but the higher blood glucose peaks are, the more hungry the subjects are prior the subsequent meal. Therefore, it would be expected that the satiating effect of the SSM would disappear faster and that hunger would reappear sooner. Evidence for this was shown in that, despite the lack of difference in the number of meal requests or intermeal intervals between the treatments, the subjects felt more satiated with SSM+ (not different from the SM results) compared to SSM (different from the SM results) before ingestion of the third meal in the afternoon. This may indicate that the decreased satiety feeling with the unsupplemented semisolid meal will occur later during the day. Although there was no difference in general appetite ratings and energy intake during the testing day in these subjects, determination of appetite in a larger group of subjects at home indicated that GG added to the same semisolid meal allowed a reduced energy intake but prevented an increase in appetite; this increase was present in the unsupplemented semisolid meal and in the solid meal (Kovacs *et al*, 2001). These results are in line with previous findings that one-week of GG supplementation effectively reduces hunger and increases satiety at a low energy intake level (Pasman *et al*, 1997). The prevention of increased appetite may improve compliance to an energy-restricted diet (Astrup *et al*, 1990) and subsequently increase the chances of successful weight loss or prevention of weight gain. On the contrary, Heini *et al*, (1998) did not find any effect of 1 week of GG supplementation during energy restriction on appetite and other measured parameters (blood concentrations of glucose, insulin, leptin, and resting

**Table 3** Area under the curve (AUC) (mm/h) from the following 100 mm VAS ratings over 16 h (baseline, before and after breakfast, lunch, snack and dinner)

	SSM+	SSM	SM	P
Hunger	364 $\pm$ 28	381 $\pm$ 28	336 $\pm$ 28	NS
Appetite	368 $\pm$ 30	414 $\pm$ 28	345 $\pm$ 30	NS
Anticipated food intake	427 $\pm$ 32	439 $\pm$ 28	370 $\pm$ 36	NS
Desire to eat	361 $\pm$ 25	402 $\pm$ 28	338 $\pm$ 30	NS
Fullness	306 $\pm$ 24	256 $\pm$ 20	292 $\pm$ 19	NS
Satiety	341 $\pm$ 24	294 $\pm$ 25	321 $\pm$ 19	NS
Thirst	330 $\pm$ 29	313 $\pm$ 30	308 $\pm$ 23	NS

Values are mean  $\pm$  s.e.

SSM+ = semisolid meal with guar gum; SSM = semisolid meal; SM = solid meal.

Statistical significance was determined by an analysis of variance for repeated measures (ANOVA). NS = not significant.

quotient), with the exception of cholecystokinin. In contrast to the form of GG used in our study, the GG they used has no viscosity effect. However, this does not explain the discrepancy with the results of Pasman *et al* (1997), as they used the same type of fiber and the same dosage, for a similar duration. Significant differences were found in blood glucose and insulin, as determined by occasional blood sampling, between the unsupplemented semisolid meal and the solid meal at certain time points. In contrast, no difference was found in blood glucose and insulin profile or in appetite ratings between the supplemented semisolid meal and the solid meal, although the solid meal is expected to have a slower gastric emptying and intestinal transit. In this respect, the supplemented semisolid meal was found as efficacious as the solid meal in order to prevent rapid changes in the blood glucose profile, which may be responsible for a quicker appearance for hunger and/or a shorter interval between meals. However, the semisolid meal was found to be much more convenient compared with the solid meal that the subjects had to prepare themselves. The use of a ready-to-eat semisolid meal would therefore result in better compliance to an energy-restricted diet in the long term compared to a self-prepared solid meal (Kovacs *et al*, 2001).

The inability of the subjects to reliably estimate clock time at the end of the testing day implies that hunger ratings and meal requests were the results of internal, physiological signals and not of external time cues.

It is concluded that SSM+ and SM showed a more moderate change in blood glucose, as determined by continuous on-line glucose monitoring and that this may have caused the relative increase in satiety in the afternoon before ingestion of the third meal. However, all three treatments had similar effects on general appetite, energy intake and meal patterns. Therefore, we conclude that the addition of guar gum to a low-energy semisolid meal prevented reduction of satiety and moderated changes in blood glucose, while maintaining the advantage of a ready-to-eat meal.

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